EFFECT OF MENTAL STRESS ON HEART RATE, BLOOD PRESSURE, AND CORONARY ARTERIAL DIAMETER

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We studied the effect of mental stress on heart rate (HR), blood pressure (BP), and coronary arterial diameter in 11 patients who underwent diagnostic cardiac catheterization. For each subject, 75 second duration continuous recording of BP and HR was performed at baseline and during performance of an emotionally arousing speech about undesirable personal characteristics. Quantitative coronary angiography of left coronary artery segments was performed at baseline and 75 seconds into the speech task. A senior angiographer, blinded with respect to (baseline or speech task) timing of angiography, performed the arterial measurements.

Compared with baseline, the speech task was associated with significant increases in mean HR (16 beats/minute, p=0.006), systolic BP (26 mmHg, p=0.004), and diastolic BP (14 mmHg, p=0.02). The average diameter of every coronary artery segment decreased with mental stress. Observations of 67 coronary artery segments demonstrated a significant decrease in mean diameter (0.32 mm, p=0.001). No relationship between change in coronary diameter and change in HR, BP, or extent of overall fixed coronary obstructive disease was observed.

Our data from patients undergoing coronary angiography suggest that mental stress (speech task) results in significant increases in mean HR and BP, and significant decrease in mean coronary arterial diameter.

IMPROVED HEMODYNAMICS AFTER SURGICAL WEIGHT LOSS IN PATIENTS WITH MORBID OBESITY

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The hemodynamic (HD) impact of morbid obesity after surgical weight (wt) loss was assessed in 28 morbidly obese patients (pts) without evidence of underlying cardiac disease, hypoxemia, diabetes mellitus, or hypertension. There were 23 females and 5 males aged 20-59 years with a mean wt of 140 ± 5.4 kg (±SEM). All underwent right heart HD studies including creation of a ventricular function curve for each pt by measuring HD at both low and high filling pressures using saline load or sublingual nitroglycerin as needed. Eleven pts agreed to a similar HD restudy 6 months after weight loss surgery. Basal resting (pre-surgical) HD in the whole group revealed a direct correlation between percentage over ideal body wt (POW) and mean pulmonary artery pressure (MPAP) ($r=0.5$, $P \leq 0.005$) and a negative correlation between POW and stroke volume ($r=0.8$, $p \leq 0.005$). Six months post-surgery, there was a significant reduction in POW ($p \leq 0.0005$) and MPAP ($p \leq 0.05$). The mean slope of the line comparing low and high filling pressure cardiac index pre-surgery was - 0.08 ± 0.25. After surgery there was a significant increase in the slope of the line to + 0.15 ± 0.16 with ($p \leq 0.04$). In conclusion, this study confirms the relationship between elevated wt and abnormal cardiac function even in pts without evidence of underlying cardiac disease. Furthermore, abnormal ventricular function apparent during obesity is reversible following surgical weight loss, providing evidence for a myopathy of obesity which is reversible with wt loss.

ALTERATIONS IN LEFT VENTRICULAR RELAXATION DURING ATRIOVENTRICULAR PACING IN MAN.

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This study was undertaken to determine if the asynchronous left ventricular (LV) contraction-relaxation sequence produced by ventricular pacing alters LV diastolic function. Accordingly, LV micromanometer pressures were recorded in 25 pts during atrial and AV sequential pacing at a heart rate 10-15 beats above resting sinus rate. Pressures were digitized on-line using a computer programmed to calculate peak negative LV dP/dt and the time constant of LV relaxation (T).

Patients were divided into 2 groups according to LV systolic function. Ejection fraction was 0.68 ± 0.08 in group 1 and 0.47 ± 0.08 in group 2 ($p<0.0001$). Heart rate, LV peak systolic pressure, LV end-systolic pressure, and LVEDP remained constant during atrial and AV sequential pacing. Results are as follows:

	neg dP/dt (mmHg/s)		T (ms)	
	atrial	AV	atrial	AV
Group 1 (n=10)	1507±200	1424±187	48±11	51±11
Group 2 (n=15)	1358±333	1222±240	59±8	71±15

Changes in peak negative dP/dt and T during AV sequential pacing were not significant in group 1. In group 2, peak negative dP/dt decreased ($p<0.0005$), and T increased ($p<0.0001$) significantly during AV sequential pacing. Furthermore, the percent increase in T correlated with increased LV wall thickness ($r=0.77$) and systolic pressure ($r=0.78$). Thus, these data establish that AV sequential pacing impairs left ventricular diastolic relaxation in patients with baseline abnormalities of left ventricular systolic and diastolic function.

WEIGHT LOSS AFFECTS VASCULAR REACTIVITY IN OBESE HYPERTENSIVES.

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Although blood pressure has been found to decrease with weight loss, the mechanism of this hypotensive effect is unclear. We examined the effects of a reduction in body weight on resting blood pressure (BP), heart rate (HR), forearm blood flow (FBF) and vascular resistance (FVR) as well as the response of these parameters to isometric hand grip (HG), mental arithmetic (MA) and forearm ischemia (ISCH), in 10 obese (130-170% of ideal body weight), moderately hypertensive (140-190/90-110) subjects. A 1 wk control (CON) period was followed by a 9 wk period of caloric restriction (WtL). The average weight loss was 10±2 kg. During the next 4 weeks food intake was adjusted to maintain weight. Blood pressure dropped significantly during WtL (12/10mm Hg, $p<0.05$) and remained reduced during maintenance. Resting FBF and HR fell with WtL (5.9 ± 0.5 to 3.3 ± 0.7; 67 ± 3, to 60 ± 3: $p<0.01$ for both), consistent with prior reports of decreased cardiac output with weight loss. However, while the response of BP and HR to HG, MA, and ISCH did not differ after WtL, WtL significantly altered the response of FBF and FVR.

	HG		MA		ISCH	
	CON	POST WtL	CON	POST WtL	CON	POST WtL
FBF[t]	27±10	86±19*	27±14	92±14**	582±86	993±138*
FVR[t]	3±9	-22±8**	-9±9	-42±7**	-81±3	-92±1*

t=expressed as percent increase over resting value
*$p<0.05$ **$p<0.01$ vs PRE WtL

Thus, although blood pressure falls with WtL vascular resistance may rise, limiting the beneficial effect.